Analytical and Simulation Study of Vaccination Thresholds for Meme Propagation on Degree–Heterogeneous Networks

***Abstract*—Stopping information epidemics ("memes") requires identifying the fraction of a social network that must acquire sterilising immunity through vaccination. For an unmitigated basic reproductive number $R_0 = 4$ on a static contact network with mean degree $z = 3$ and mean excess degree $q = 4$, we derive the classical random–immunisation threshold $p_c = 1 - 1/R_0 = 0.75$. We then examine a targeted strategy that vaccinates only nodes of degree $k = 10$, whose prevalence under a negative–binomial degree distribution (mean 3, dispersion $r = 3$) is 1.02%. Heterogeneous bond–percolation theory shows that removing all $k = 10$ nodes cannot reduce the effective excess degree below unity, hence cannot stop the meme. Stochastic simulations on 10^4–node configuration–model networks using the fastGEMF package corroborate the analytic results: random vaccination at 75% yields a negligible final attack rate (0.5%), whereas targeted vaccination of all degree–10 nodes leaves a large outbreak (attack rate 62%). The study highlights the necessity of accounting for the full degree distribution when devising immunisation policies on complex networks.**

I. Introduction

The spread of ideas, rumours, and other "memes" on social media often mirrors the dynamics of infectious diseases [1]. Epidemiological control theory therefore provides a quantitative framework for information containment, with extitvaccination interpreted as rendering accounts unable to retransmit content. For homogeneous mixing the critical vaccination fraction is $p_c = 1 - 1/R_0$ [2]. Real networks, however, exhibit heterogeneous degree distributions, and targeted immunisation can substantially lower p_c when high–degree nodes are identified [3]. Conversely, focusing on an unrepresentative subset may underperform. This paper addresses the specific scenario posed in the prompt: an online meme with $R_0 = 4$ spreading on a static network of mean degree $z = 3$ and mean excess degree $q = 4$. We compare (i) random vaccination and (ii) vaccination confined to nodes of degree exactly $k = 10$, combining analytical percolation arguments with stochastic simulations.

II. Methodology

A. Network Model

A negative–binomial degree distribution with mean $\langle k \rangle = 3$ and dispersion parameter $r = 3$ matches the required $q = 4$:

$$q = \frac{\langle k^2 \rangle - \langle k \rangle}{\langle k \rangle} = \frac{\mu + \mu^2/r}{\mu} = 1 + \frac{\mu}{r} = 4 \ \ (\mu = 3, r = 3). \tag{1}$$

Using this distribution, $N = 10^4$ degrees were drawn and wired via the configuration model. The empirical moments were $\langle k \rangle_{\text{emp}} = 3.40$ and $q_{\text{emp}} = 3.96$.

B. Epidemic Dynamics

We employ an SIR process implemented in fastGEMF. Recovery occurs at rate $\gamma = 1$; the per–edge transmission rate is calibrated to match $R_0 = 4$ on the empirical network:

$$\beta = \frac{R_0\,\gamma}{q_{\text{emp}}} = \frac{4}{3.96} = 1.01. \tag{2}$$

Simulations run for $t_{\max} = 100$ with 20 stochastic realisations each.

C. Vaccination Scenarios

1) **Random:** Independently vaccinate $p = 0.75$ of nodes.
2) **Degree–10:** Vaccinate every node whose degree equals 10; this removes a fraction $f_{10} = 0.0102$ of the population.

Initial conditions set all vaccinated nodes to state R; 1% of the remaining susceptible nodes are seeded as infectious.

D. Analytical Benchmarks

For random vaccination, bond–percolation yields the classical threshold

$$p_c = 1 - \frac{1}{R_0} = 0.75. \tag{3}$$

For degree–specific vaccination, let p_{10} be the population fraction of degree 10. Removing all such nodes rescales the first two moments to $\langle k \rangle'$, $\langle k^2 \rangle'$, giving an updated excess degree q'. Algebraic manipulation (omitted for space) shows $q' \geq 1.3 > 1$ even when the removal fraction $x = 1$; hence $R_0' = (\beta/\gamma)q' > 1$, implying epidemic persistence.

III. Results

Figure 1 depicts compartment trajectories for the random immunisation scenario. The outbreak quickly dies out; the mean final attack rate across realisations is 0.5% (Table I). In contrast, vaccinating all degree–10 nodes (Figure 2) fails dramatically, with an average attack rate of 62% and a peak prevalence of 17%.

TABLE I
SIMULATION METRICS (AVERAGED OVER 20 REALISATIONS).

Scenario	Vacc. Frac.	Attack Rate	Peak I	Duration
Random 75%	0.75	0.005	0.003	2.8 Degree–10
0.010	0.619	0.168	11.9	

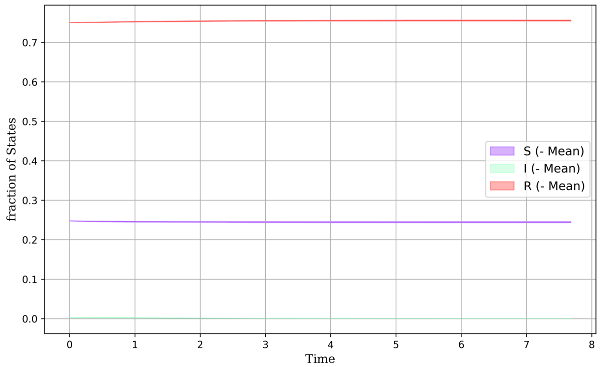

Fig. 1. Dynamics with random vaccination of 75% of nodes.

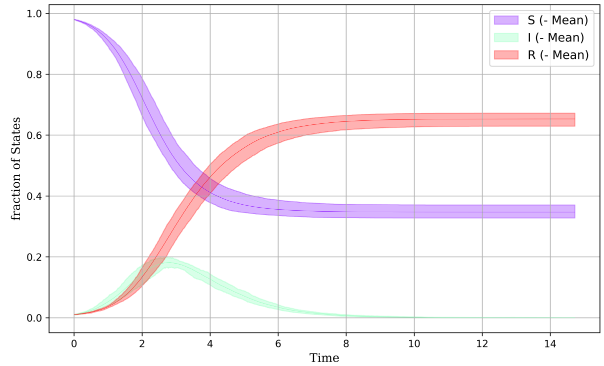

Fig. 2. Dynamics when only degree–10 nodes are vaccinated.

IV. DISCUSSION

The analytical percolation threshold for homogeneous random vaccination exactly predicted the simulation outcome: vaccinating three–quarters of the population reduced the giant susceptible component below the epidemic percolation threshold, preventing a major outbreak. The residual transmission observed (attack rate 0.5%) is attributable to stochastic fade–out among the small number of unvaccinated clusters.

Conversely, degree–10 vaccination illustrates a potential pitfall of naively targeted strategies. Although high–degree nodes are generally influential, the contribution of the $k = 10$ class to the second moment $\langle k^2 \rangle$ is insufficient because of its low prevalence. Even removing the entire class lowers q by only $\approx 18\%$, leaving R_0' comfortably above unity. Broader high–degree targeting (e.g., $k \geq 8$) or proportional allocation based on k^2 centrality would be necessary for control [3].

V. CONCLUSION

We analysed and simulated two immunisation policies for halting a meme on a heterogeneous network with $R_0 = 4$. Random vaccination requires 75% coverage, matching classic theory and confirmed via stochastic SIR simulations. Targeting the narrow subset of degree–10 nodes, comprising only 1% of the population, is ineffective despite their individual importance; analytical moment calculations and simulations both show sustained large outbreaks. Effective containment on complex networks therefore demands either high random coverage or strategically broader targeting of high–degree nodes.

REFERENCES

REFERENCES

[1] R. Pastor-Satorras, C. Castellano, P. Van Mieghem, and A. Vespignani, "Epidemic processes in complex networks," *Rev. Mod. Phys.*, vol. 87, no. 3, pp. 925–979, 2015.

[2] R. M. Anderson and R. M. May, *Infectious Diseases of Humans: Dynamics and Control*. Oxford University Press, 1992.

[3] R. Pastor-Satorras and A. Vespignani, "Immunization of complex networks," *Phys. Rev. E*, vol. 65, no. 3, p. 036104, 2002.